EXHIBIT 116

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Restatement of the Law -- Torts
Restatement (Third) of Torts: Liability for Physical Harm (Tentative Drafts) FNa
Current through August 2007

Chapter 5. Factual Cause

§ 28. Burden Of Proof

Tentative Draft No. 5

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(a) Subject to Subsection (b), the plaintiff has the burden to prove that the defendant's tortious conduct was a factual cause of the plaintiff's physical harm.

(b) When the plaintiff sues all of multiple actors and proves that each engaged in tortuous conduct that exposed the plaintiff to a risk of physical harm and that the tortious conduct of one or more of them caused the plaintiff's harm but the plaintiff cannot reasonably be expected to prove which actor caused the harm, the burden of proof, including both production and persuasion, on factual causation is shifted to the defendants.

Comment:

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e. Reasonable medical or scientific certainty. Experts must hold their opinions with some degree of certainty for them to be admissible. To an expert witness, virtually any proposition may be "possible," but the law demands proof by a preponderance of the evidence in civil cases. In an effort to screen expert opinions that are speculative, some courts have employed a requirement that an expert testify that an opinion is held to a "reasonable degree of medical [or scientific] certainty" for it to be admissible. This phrase implies a standard different from the preponderance requirement, suggests reliance on medical or scientific standards for proof, and seems to impose a high threshold for the opinion to be admissible. Some courts avoid the last-mentioned implication by employing instead a requirement that the expert testify that an opinion is held to a "reasonable medical [or scientific] probability."

Requiring an expert to state that an opinion is held to a medical or scientific certainty is problematic because the medical and scientific communities have no such "reasonable certainty" standard. Thus for an expert to understand this standard, meaning must be provided by the attorney who hired the expert, by the expert's imagination, or by some other source outside the legal system. The implication that the reasonable-certainty standard requires something more than the preponderance standard is belied by courts that have provided a definition of or explained the phrase--the vast majority of those courts state that the standard is equivalent to the usual preponderance requirement.

There is a troubling inconsistency in imposing a higher threshold for the admissibility of expert testimony

In tort cases in which the defendant's tortious conduct is clear, many courts are lenient about the plaintiff's proof of causation, especially if the plaintiff has done all that is reasonably possible by way of gathering and presenting evidence of causation. Perhaps the best example of easing the plaintiff's burden on factual causation is found in those courts that have adopted a presumption of causation in negligence per se cases. Other courts have adopted a presumption of causation when the defendant fails to warn or provides an inadequate warning in products-liability cases. The use of presumptions in negligence per se and warnings cases is by no means universal, but it does reflect some courts' willingness to adapt the burden of proof depending on the type of tortious conduct and the difficulties of proof the plaintiff faces.

Beyond negligence per se and warnings cases, in unusual circumstances in addition to those in Subsection (b), courts may shift the burden of proof on causation to defendants. Courts have shifted the burden of proof on the aspect of causation that requires identification of the actor who committed the tortious conduct--there being adequate evidence that the tortious conduct caused the plaintiff's harm. These rare cases are characterized by a close relationship among the actors who potentially caused the other's harm, the actors having superior knowledge of the relevant circumstances, and the person harmed having no reasonable prospect for obtaining evidence of causation.

In some cases the specific facts of the defendant's tortious conduct and its relationship to the harm to the plaintiff may be sufficient to justify a reasonable inference of causation. Yet it is clear that the fact of the defendant's tortious conduct and harm to a plaintiff within the scope of the risk created by that conduct cannot alone be sufficient in all cases to permit an inference of causation. As a matter of scope of liability, defendant's tortious conduct must increase the risk of harm to the plaintiff. See § 29. If nothing further were required, other than adequate evidence of tortious conduct, plaintiff's burden of production on causation would always be satisfied. Thus, only when the tortious conduct reasonably could be found, after the fact, to have increased the risk of harm to a greater extent than the risk posed by all other potential causes would an inference from tortious conduct alone be permissible. Of course, this standard underscores the critical role of the identification of and elimination vel non of those other potential causes.

In the end, the line between permissible inference for the jury and impermissible speculation is one that must be determined based on the specific facts of the case and the jurisdiction's general procedural approach to the allocation of decisionmaking authority between judge and jury.

c. Toxic substances and disease

(1) Introduction. Cases involving toxic substances often pose difficult problems of proof of factual causation. These problems can also arise in cases involving activities that may cause disease, such as continued repetitive motion. Sometimes it is difficult to prove which defendant was connected to the toxic agent, see Comment *o*, or whether an adequate warning would have prevented the plaintiff's harm. See Comment *b*. The special problem in these cases, however, is proving the connection between a substance and development of a specific disease. In all of these cases, the requirement to prove factual causation remains the same; the plaintiff must prove it by a preponderance of the evidence, and the standards for factual causation set forth in §§ 26-27 continue to apply.

In most traumatic-injury cases, the plaintiff can prove the causal role of the defendant's tortious conduct by observation, based upon reasonable inferences drawn from everyday experience and a close temporal and spatial connection between that conduct and the harm. Often, no other potential causes of injury exist. When a passenger in an automobile collision suffers a broken limb, potential causal explanations other than the collision are easily ruled out; common experience reveals that the forces generated in a serious automobile collision are **cap-**

able of **causing** a fracture. By contrast, the causes of some **diseases,** especially those with significant latency periods, are generally much less well understood. Even known causes for certain **diseases** may explain only a fraction of the incidence of such **diseases,** with the remainder due to unknown causes. Causal agents are often identified in group (epidemiologic) studies that reveal an increase in **disease** incidence among a group exposed to the agent as compared to a group not exposed. Biological mechanisms for **disease** development--i.e., a series of causally linked physiological changes from exposure to **disease** developments--are frequently complicated and difficult to observe. Science continues to develop a better understanding of the biological steps in the development of **diseases,** but current knowledge in this respect is considerably more modest than for traumatic injury. As a consequence, courts in toxic-substances cases often must assess various alternative methods proffered with regard to factual **causation.**

Over the past several decades, courts have devoted a great deal of energy to the issue of **causation** in toxic-tort cases. **Causation** is a question of fact normally left to the jury, unless reasonable minds cannot differ. Appellate or trial-court review of jury findings affects the allocation of power between judges and juries. Until the early 1980s, a qualified expert witness's opinion that a toxic agent was a factual cause of the plaintiff's disease was treated as sufficient evidence. A few celebrated cases and case congregations, such as the Agent Orange and Bendectin litigations, led some courts to distrust juries' ability to resolve cases based on conflicting general expert-opinion evidence. Courts began to scrutinize the scientific evidence employed and to examine carefully the bases for an expert's opinion on factual causation. Some courts then tried to develop bright-line rules based on science for adequate proof of factual causation. The high water mark for this overreliance on scientific thresholds occurred in the Bendectin litigation when one court announced a blanket rule that a plaintiff could not make out a sufficient case without statistically significant epidemiologic evidence.

These courts may be relying on a view that "science" presents an "objective" method of establishing that, in all cases, reasonable minds cannot differ on the issue of factual causation. Such a view is incorrect. First, scientific standards for the sufficiency of evidence to establish a proposition may be inappropriate for the law, which itself must decide the minimum amount of evidence permitting a reasonable (and therefore permissible) inference as opposed to speculation that is not permitted. See Comment *b*. Second, scientists report that an evaluation of data and scientific evidence to determine whether an inference of causation is appropriate requires judgment and interpretation. Scientists are subject to their own value judgments and preexisting biases that may affect their view of a body of evidence. There are instances in which although one scientist or group of scientists comes to one conclusion about factual causation, they recognize that another group that comes to a contrary conclusion might still be "reasonable." These scientists' views reflect their scientific experience outside the courtroom. They may have different views about specific instances of conflicting scientific testimony in a courtroom. Judgments about causation may also be affected by the comparative costs of errors, as when caution counsels in favor of declaring an uncertain agent toxic because the potential harm it may cause if toxic is so much greater than the benefit foregone if it were not introduced. Courts, thus, should be cautious about adopting specific "scientific" principles, taken out of context, to formulate bright-line legal rules or conclude that reasonable minds cannot differ about factual causation.

This Comment is necessarily general. It addresses how methods of proof for traumatic injuries and for diseases may differ. Toxic-substance cases often involve statistical and group-based scientific studies that courts seldom confronted when the Restatement Second of Torts was published. Toxic agents and the diseases they cause differ, and methods of proof may vary accordingly. The law continues to evolve as courts are confronted with a variety of different circumstances related to different toxic substances, different disease, and the varieties of available evidence.

Scientific methods may advance in the future to better facilitate causation determinations for individuals, thereby obviating the need for statistically based group studies. While such techniques are largely unavailable today, dramatic advances in microbiology, genetics, and related fields have been made. These developments may produce new forms of evidence to which courts will adapt legal treatment of proof of causation.

Proof of causation often involves the admissibility of expert-witness opinions. Admissibility is governed by the law of evidence, and nothing in this Comment addresses that law. However, admissibility cannot be determined without reference to the substantive law. Moreover, courts may be required to examine scientific evidence when it is offered to prove agent-disease causation. That examination may occur either in the admissibility determination or in the determination whether the evidence is sufficient to meet the burden of production. These usually are separate issues and are subject to different legal standards. Courts, however, sometimes conflate these issues in the process of determining whether there is an adequate basis for an expert's opinion. When courts collapse the sufficiency determination into the question of the admissibility of an expert's testimony no subsequent inquiry into sufficiency is necessary, and the appropriate weight to give to an expert's opinion once it is deemed admissible is for the factfinder. The requirement of causation, the elements of agent-disease causation that are sometimes required when group studies are employed as proof, and the sufficiency of the evidence to meet the burden of production on causation are matters of substantive tort law, and they are addressed in the Restatement.

Most causation issues are resolved under the "but-for" standard for factual cause. See § 26. The plaintiff must prove by a preponderance of the evidence that, but for the defendant's tortious conduct with respect to the toxic substance, the plaintiff would not have suffered harm. When group-based statistical evidence is proffered in a case, this means that the substance must be **capable** of **causing** the **disease** (**"general causation"**) and that the substance must have **caused** the plaintiff's **disease** (**"specific causation"**). In other cases, when group-based evidence is unavailable or inconclusive, and other forms of evidence are used, the general and specific **causation** issues may merge into a single inquiry. In any case, plaintiff's exposure to the toxic agent must be established.

Thus, courts often address "exposure," "general **causation,**" and "specific **causation.**" Nevertheless, these items are not "elements" of a plaintiff's cause of action, and in some cases may not require separate proof. So long as the plaintiff introduces admissible and sufficient evidence of factual **causation** the burden of production is satisfied. A court in a particular case may conclude that reasonable minds cannot differ about proof of factual **causation** under the general test *because* reasonable minds cannot differ on whether the plaintiff was exposed to the agent, whether the agent is generally **capable** of **causing** the **disease**, or whether the agent **caused** the plaintiff's **disease** in the specific case. These categories function as devices to organize a court's analysis, not as formal elements of the cause of action.

(2) Exposure to the agent. In evaluating factual **causation,** one issue that may arise is whether the plaintiff was exposed to the substance. Three primary means of exposure to toxic substances include inhalation, absorption, and ingestion, but others exist, such as injection or a fetus's transplacental exposure to agents in the mother's body. Often the method of exposure is critical to the type or extent of risk.

Exposure is frequently disputed in occupational-disease cases and hazardous-waste cases, while it is less often an issue in pharmaceutical cases. Proof of exposure may entail relatively straightforward historical facts, such as the presence of asbestos at the plaintiff's workplace or whether the plaintiff took a prescribed drug, or it may require complicated scientific evidence, such as dispersion modeling, to determine how and where the substance was transported. The latter form of evidence is often required in airborne-or groundwater-pollution cases. The intensity and duration of exposure (the "dose") affects the magnitude of the risks posed and the likelihood

evidence on specific causation when group-based studies are involved. Properly understood and applied, this analytical framework provides a reasonable basis for determining specific causation in the absence of more particularistic evidence about the cause of plaintiff's disease.

Courts have reasoned that when a group study finds that exposure to the agent causes an incidence in the exposed group that is more than twice the incidence in the unexposed group (i.e., a relative risk greater than two) the probability that exposure to an agent caused a similarly situated individual's disease is 50 percent. Accordingly, courts generally hold when there is group-based evidence finding that exposure to an agent causes an incidence of disease in the exposed group that is more than twice the incidence in the unexposed group, the evidence is sufficient to satisfy the burden of production and permit submission of specific causation to a jury. In such a case, the factfinder may find that it is more likely than not that the substance caused the particular plaintiff's disease. The propriety of this "doubling" reasoning depends on group studies identifying a genuine causal relationship and a reasonably reliable measure of the increased risk. Courts appropriately have permitted expert witnesses to testify to specific causation based on the logic of the effect of a doubling of the risk and other considerations explained below that modify the probability of causation for a particular individual.

Additional considerations affect the propriety of determining the probability of specific causation based on the outcome of a group-based study. Depending on the state of the evidence about these additional matters, they may bear either on the sufficiency determination by the court or be relevant to the jury's determination. Thus, the extent to which the group-study outcome reflects the increased risk to the plaintiff depends on the plaintiff's similarity to those included in the group study. Relevant differences include whether: (a) the plaintiff was exposed to a comparable dose; (b) the plaintiff was not differentially exposed to other potential causes of the disease; and (c) the plaintiff has individual characteristics that might also bear on the risk of disease, such as age, gender, or general health, comparable to those in the study group.

The likelihood that an agent caused an individual's disease may be refined when there are independent, alternative known causes of the disease. The underlying premise is that each of these known causes is independently responsible for some proportion of the disease in a given population. Eliminating one or more of these as a possible cause for a specific plaintiff's disease increases the probability that the agent in question was responsible for that plaintiff's disease. Courts frequently refer to the elimination of other known causes for a plaintiff by employing the medical terminology of "differential diagnosis." The logic is sound, but the terminology and attribution are not. Assessing whether other causes can be ruled out (or in) as potential causes of a plaintiff's disease can provide probative evidence of specific causation. This technique is more accurately described as a "differential etiology." It is most useful when the causes of a substantial proportion of the disease are known. Then, the presence (or absence) of these causes for the specific plaintiff affects the probability that the agent in question caused the plaintiff's illness. When the causes of a disease are largely unknown, however, differential etiology is of little assistance. Evidence

about biological mechanisms may also alter the likelihood that exposure to the substance caused plaintiff's disease, either by ruling out other known causes or by explaining why the suspected agent is a more likely cause of the disease than others.

For all of these reasons, any judicial requirement that plaintiffs must show a threshold increase in risk or a doubling in incidence in a group study in order to satisfy the burden of proof of specific causation is usually inappropriate. So long as there is adequate evidence of general causation, courts should permit the parties to attempt to show, based on the sorts of evidence described above, whether the plaintiff's disease was more likely than not caused by the agent. Depending on the other factors detailed above, an increase of the incidence of disease less than a doubling may be sufficient to support a finding of causation, while in another case, even an increased incidence greater than two may not be sufficient. When the sufficiency of the evidence to meet the burden of production is at issue, courts should consider all of the evidence that bears on the matters discussed above and determine whether, in light of the general standard for sufficiency discussed in Comment *b*, the evidence would permit a reasonable jury to find that plaintiff's disease more probably than not was caused by exposure to the agent.

In most instances, differential etiology is not an appropriate technique for proving general causation. Nevertheless, in some limited circumstances courts have found that plaintiffs met their burden of proof of agent-disease causation without separate proof of general causation. Factors such as a good biological-mechanism explanation of how the agent could have caused the plaintiff's disease, a differential etiology ruling out other known causes, a reasonable explanation for the lack of general-causation evidence (and no contrary evidence of an absence of general causation), a short latency period and acute response, and the appropriate disease response to dechallenge (removal from exposure) and rechallenge (reexposure) to the agent, if combined and consistent, provide a persuasive basis for excusing the plaintiff from providing other proof of general causation.

(5) Multiple exposures and synergistic interactions. In some cases, a person may be exposed to two or more toxic agents, each of which is known to be capable of causing (general causation) the person's disease. The two agents may operate independently, in which case the incidence of disease in a group exposed to both will be additive--the excess incidence due to the first agent along with the excess incidence due to the second agent. Cases such as these present a relatively straightforward application of the principles set forth in Comment *c(4)*. If the toxic agents are attributable to the tortious conduct of separate actors, courts then face the question whether to apply the rule developed for multiple exposures in asbestos cases. This rule permits finding each actor's asbestos products to which the person was exposed to be a factual cause of the person's disease. See § 27, Comment *g*. Alternatively, courts might employ the traditional rule, requiring proof of which of the multiple exposures was a cause of the harm. At least where the biological mechanism by which disease develops is unknown, the asbestos rule is quite analogous and attractive as a means for adapting proof requirements to the available scientific knowledge. Apportionment of liability among those actors held liable is based on the comparative-responsibility rules in Restatement Third, Torts: Apportionment of Liability §§ 1-25. The alternative--the more traditional